Degenerative Lesions of the Shoulder as a Cause of Pain

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PAINFUL degenerative lesions of the shoulder are ordinarily associated with advancing years. They include changes in the tendons, shoulder capsule and the joint cartilage. Terms such as tendinitis, bursitis, periarthritis, arthritis, and others, are given to these lesions. The symptoms are frequently initiated by trauma, even though minor. Degenerative changes are the major cause of prolonged disability beyond the second and third decades.

The shoulder joint is the most mobile in the body. It depends on its muscle and tendinous support for a large share of its stability. The rotator cuff and its spinatus tendons must travel a long way during resisted abduction of the arm, while they are under pressure between the head of the humerus and the acromion. The long head of the biceps is similarly situated medial to the greater tuberosity, extending medialward over the humeral head within the joint capsule to its insertion on the upper pole of the glenoid.

The classical description of the lesions of the shoulder joint which lead to pathological rupture, as well as to calcification of the rotator cuff as given by Codman [2] is clear and concise. The earliest gross changes which give evidence of degeneration are loss of the normal elasticity of this tissue. On microscopic examination, this change is evidenced by a hyalin degeneration in the collagens of the tendon fibers. This degeneration is followed by fibrillation or separation of the individual fibers which then become more easily vulnerable to the trauma of movement. If enough of these individual changes have occurred in the substance of a tendon, there may be various degrees of separation in local areas of these thickened, swollen, degenerated tendon fibers. As they have lost their normal elasticity, additional trauma may break them up into small pieces so that they eventually become what have been termed "rice bodies." These small accumulations of loose degenerated tendon fibers may undergo a process of healing, or gradually enlarge as other fibers are added to the original mass. Although the material in these cavities appears grossly at operation as an accumulation of pus, it is sterile on culture. If trauma is not sufficient to produce major ruptures of the tendon. this degenerative mass of material may become calcified. The mechanism for this change is not known. The degenerative process is usually reversed when a rupture or tear occurs in some overlying tendon fibers so that the material may be thrown out into more vascular tissue such as the subdeltoid or the subacromial bursa. Granulation tissue then invades the cavity in the healing process. When this material is thrown out into tissue which is more vascular, the normal foreign body reaction takes place. The inflammatory reaction around this area when it is small can quickly repair these tissues. If, however, a tear should occur, and a large amount of debris is thrown into the subdeltoid bursa, or if calcareous material is thrown out into the subdeltoid bursa, the inflammatory reaction which takes place will be acute. Early calcareous deposits are at times the seat of inflammation. Prior to rupture there may be marked pressure increase within the sac. This results in acute pain which will be aggravated by any type of treatment or pressure that increases deep engorgement or congestion.

The calcareous material, at first liquid and milky, gradually becomes dry and chalky in the chronic cases. The degenerated bands or strands are easily seen when the bursa is opened. When the dry, chalky material is curetted from the various small pockets, the edges are dry, rough and avascular, until the base and margins have been cut away. These margins have the gross appearance of fibrillated degenerated tendon. It may be necessary to cut through the cortical plate of the bone at the base to get free bleeding.

The joint cartilage changes are at first swelling and softening, and later fibrillating and thinning, especially in those shoulders that have soft tissue degeneration. The subchondral bony plate may become hard and sclerotic.

The degenerative changes previously enumerated are, in the author's opinion, primarily metabolic, with heredity an important determinant. Just as gray hair appears earlier in the offspring of certain parents, so degenerative tendencies also are transmitted. These tendencies may be manifest through a gradual slowing down of circulation to part of or all the body tissues with advancing years. Since tendons and joint cartilage are poorly nourished as compared with muscle, they are the first to undergo the changes which anoxia brings about.

Trauma plays an important part in many cases, in bringing on the acute changes, but it is not the sole cause, for even greater trauma in younger people produces only brief disability or none at all. If trauma were the major consideration, then why should we see more women than men disabled? And why are the slender, small boned, poorly muscled so frequently affected, even though they have been doing almost sedentary work? It is noteworthy that, in the author's experience, shoulder lesions in patients in this category have been the most resistant to treatment, and convalescence has been slower. Trauma may be caused by bacterial or other toxins which can sharply accelerate tissue senescence. Such traumata seem quite pronounced in what we choose to call rheumatoid arthritis.

Patients, usually over 30 years of age and most frequently 40 to 50, give a history of either a slowly progressive ache in the shoulder without trauma, or of a rather minor injury followed by a lull of eight to twenty hours of relative comfort before the acute symptoms begin. When the patient is acutely affected

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he has severe shoulder pain, with an elevation of temperature to 100 degrees or 101 degrees F., pain on any arm movement and severe tenderness on pressure over the greater tuberosity. The author has seen patients with a leukocyte count as high as 16,000, although this is not common. The pain apparently is especially annoying at night. The application of heat, diathermy, for example, intensifies the pain; cold relieves it. In about one to two weeks the pain begins to subside, and the patient may begin to move the arm a little. This relief occurs, even though the only treatment is the splinting imposed by nature. McLaughlin[3] states that in a large series studied, results among patients given no treatment except sedatives were strikingly similar to those obtained in patients who were given vigorous treatment. Calcifications need not cause pain, as Bosworth[1] found that 2.7 per cent of a series of 6,000 of so-called normals had calcifications without symptoms.

In the chronic cases the patients may have severely stiff shoulders for periods of from six months to four years. Pain is not proportional to the original injury or to the degree of stiffness. A very high percentage of patients will recover near-normal range of motion unless the joint has been damaged by vigorous manipulation or too strenuous physiotherapy.

No x-ray examination is complete that does not provide a two-view projection, one view setting the greater tuberosity in relief in either internal or external rotation, the other a view at 90 degree angle from this. The early changes are seen as a decalcification or pitting of the greater tuberosity. Later this same area may be the site of calcification. The early calcifications during the "milky stage" are seen as a hazy shadow. The dry, chalky deposits are seen as sharply outlined shadows. These are sometimes seen in the subscapularis tendon as well as in the supraspinatus insertion.

The author's choice of treatment during the very acute phase is, in the absence of calcification, the application of ice caps directly to the shoulder. These are applied for 20 minutes and then removed for 20 minutes, this alternation being continued the first 24 to 48 hours, when the acute symptoms usually have subsided. If fever is present, the use of penicillin and sulfa drugs for the same period accelerates the return to normal. Later the ice caps are alternated with hot water bags or an infra-red lamp. In those patients who have visible calcifications, surgical removal will give the quickest, long-term relief.

In both instances those movements which cause pain are strictly proscribed. When the patient can move the arm even a small amount without pain he is encouraged to move it actively, but within the painless range. The airplane splint is a great help both in aiding early exercise of the shoulder and in preventing the adducted frozen shoulder which is sometimes seen. It is most important that the exercise be active and that it be done as many times a day as possible, as long as it does not result in stiffening or excessive after pain.

The same active exercise program is prescribed in the subacute cases. Any measure which will promote local increase in circulation is indicated. Application of heat (especially before exercise), alternate hot and cold applications, local infiltrations of novocain as well as stellate ganglion blocks, and many measures are useful. Because of the long time periods involved in many cases, patients are sometimes overtreated. Massage, if given carefully, will do a great deal to alleviate muscle tenderness. The excessive use of deep heat, such as diathermy or x-ray therapy, in chronic cases probably prolongs rather than accelerates the recovery rate. The clinical course appears to be self-limited for each episode. The degree of change determines the time of recovery. A great deal of our emphasis is directed to decreasing secondary reaction and lessening the trauma of moving a swollen greater tuberosity under the acromion.

Although some patients in the higher age brackets should be urged to decrease their work or change the nature of it, ordinarily they should be permitted to perform as much of their work as they can as soon as they can. This helps keep up morale—which is important in the treatment—when the progress is slow. Assurance may be given that the prognosis is excellent so far as eventual use of the joint is concerned.

The immediate results in acute cases, whether operation or conservative therapy is used, are good. Even those patients who have had tears of the rotator cuff will be able, with the aid of local novocain blocks, to start an exercise program and to keep up a surprising amount of it after the effect of the drug has worn off.

If quick repair is expected, the results in chronic cases are discouraging. Stellate ganglion blocks give rather consistent relief of pain, but this relief is more lasting in the more acute cases. When the patient is of the slender, sthenic type, time alone often seems to bring healing, although it may be three to eighteen months before satisfactory function returns.

SUMMARY

1. The pathological changes in degenerative lesions of the shoulder have been reviewed.

2. These have been given as an explanation for the frequent chronic cases seen from minor injury.

3. Acute cases are treated preferably by cold rather than heat.

4. Exercise should always be *active* and within the painless range.

5. Overtreatment and manipulations are two of the causes of prolonged disability.

6. Novocain infiltrations locally, as well as stellate ganglion blocks, are valuable adjuncts to treatment.

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REFERENCES

1. Bosworth, Boardman March: Calcified deposits in the shoulder and subacromial bursitis; a survey of 12,122 shoulders, J.A.M.A., 116:2477 (May 31), 1941.

2. Codman, E. A.: The shoulder, 1934, pp. 65-122.

3. McLaughlin, Harrison L.: Calcific deposits in the shoulder, N. Y. State J. of Med., 44:2231-2235 (Oct. 15), 1944.